Comparative Hazards of Chrysotile Asbestos and Its Substitutes: A European Perspective

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Although the use of amphibole asbestos (crocidolite and amosite) has been banned in most European countries because of its known effects on the lung and pleura, chrysotile asbestos remains in use in a number of widely used products, notably asbestos cement and friction linings in vehicle brakes and clutches. A ban on chrysotile throughout the European Union for these remaining applications is currently under consideration, but this requires confidence in the safety of substitute materials. The main substitutes for the residual uses of chrysotile are *p*-aramid, polyvinyl alcohol (PVA), and cellulose fibers, and it is these materials that are evaluated here. Because it critically affects both exposure concentrations and deposition in the lung, diameter is a key determinant of the intrinsic hazard of a fiber; the propensity of a material to release fibers into the air is also important. It is generally accepted that to be pathogenic to the lung or pleura, fibers must be long, thin, and durable; fiber chemistry may also be significant. These basic principles are used in a pragmatic way to form a judgement on the relative safety of the substitute materials, taking into account what is known about their hazardous properties and also the potential for uncontrolled exposures during a lifetime of use (including disposal). We conclude that chrysotile asbestos is intrinsically more hazardous than *p*-aramid, PVA, or cellulose fibers and that its continued use in asbestos-cement products and friction materials is not justifiable in the face of available technically adequate substitutes. *Key words*: aramid, asbestos, cancer, cellulose, chrysotile, fibrosis, hazards, PVA, substitute fibers. *Environ Health Perspect* 107:607–611 (1999). [Online 24 June 1999]
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Concern about asbestos stems from the identification in the 1930s onward of asbestosis, lung cancer, and mesothelioma in workers in the asbestos industries. In the 1960s it was recognized that the amphibole forms, mainly crocidolite and amosite, were the most hazardous in relation to mesothelioma, and their use was subsequently banned in the United Kingdom and elsewhere. More recently some countries have also banned chrysotile, and a European Union (EU) ban is currently under consideration. Although it is still used in the United Kingdom and some other member states, there has been continuing progress in replacing chrysotile with substitute fibrous materials or by alternative technology. Thus in 1975 the total amount of chrysotile imported into the United Kingdom was 191,740 metric tons; by 1997 this had fallen to 4,820 metric tons. Until its use was restricted, chrysotile was a component of numerous products including various building materials. The remaining applications are mainly in asbestos cement and friction materials.

In this paper we present reasoned scientific arguments and judgments on the substitution of chrysotile for these specific purposes; a comprehensive review of all the available data is not presented here. The paper is based on a case that was prepared for the UK Health and Safety Commission (London), and was in turn submitted to the European Commission (Brussels). The appropriate independent scientific committee of the European Commission has since adopted a broadly similar conclusion, and discussions are now under way for the issuance of a new policy statement. This paper was written from a European perspective, drawing largely from UK conditions and experiences. The conclusions, however, will doubtless be of interest and relevance in many countries worldwide.

This paper specifically addresses the main substitutes for the remaining residual uses of chrysotile, i.e., *p*-aramid, polyvinyl alcohol (PVA), and cellulose, and therefore does not cover substitute materials already widely used for thermal and sound insulation, such as glass and other man-made mineral fibers. Finally, the paper focuses only on health impacts and does not attempt a cost-benefit analysis.

Chrysotile Substitutes

The nature and uses of chrysotile substitutes have been reviewed by Hodgson (*1*). Alternatives to chrysotile have always been available, including PVC and sheet metal to replace asbestos cement, and metal gaskets and calcium-silicate insulating boards. Substitution of asbestos involves the use of other fibers in place of chrysotile, and for nearly three decades there has been a requirement for all UK asbestos users to actively seek substitutes (*2*). In the EU chrysotile has been classified as a category 1 carcinogen [Dangerous Substances Directive, 67/548/EEC; (*3*)]. In the United Kingdom this led to the Control of Asbestos at Work Regulations (*4*). The main nonasbestos fibers that are currently being exploited in the United Kingdom as substitutes for the remaining uses of chrysotile asbestos are PVA, aramid fibers, and cellulose.

Products for Which Asbestos Can Be Substituted

Asbestos-cement products. The major asbestos-cement products are profiled sheet, flat sheet, and building board, slates, pressure pipes, and molded goods. Most commonly, PVA and cellulose are used as substitutes, particularly for sheet and slates. Polyacrylonitrile (PAN) or glass fiber may also be used. PVA and PAN require the inclusion of cellulose pulp for conventional asbestos-cement manufacturing processes. High-quality cellulose has good potential as a substitute fiber. Its reinforcing properties can be improved by increasing the loading relative to that used for asbestos, or by incorporating synthetic fiber such as PVA. The temperature resistance is not as good as for asbestos cement, but can be enhanced by the addition of mica or the natural mineral wollastonite. Substitute fibers do not appear promising for pressure pipes because of strength requirements, but alternative materials may be used, e.g., unplasticized polyvinyl chloride.

Friction materials. There are three major friction products—brake linings, brake pads, and clutch facings. The composition of asbestos-based products is complex for all these applications, which have been developed to perform under extreme forces and temperatures without failing. A typical

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asbestos brake lining would be composed of 40% chrysotile with over 20 other components, including phenolic resins. The predominant substitute for chrysotile in friction products is aramid fiber, although PAN, other fibers, and some metal and semi-metallic materials are also used, often in combination.

Gaskets and sealing materials. Gaskets made from compressed asbestos fiber are widely used in turbines, compressors, and motor vehicle engines. A wide range of substitutes has been and is being developed, including aramid fiber in conjunction with other fibers such as cellulose pulp or glass fiber with various mineral fillers.

Seals include dry packings and impregnated packings. The former are used as barriers to flame spread in static sealing applications, such as around furnace and kiln doors and around floodlight lamps. Glass yarn and mineral wools are available as substitutes in these applications. Impregnated packings provide a seal between moving surfaces, e.g., in compressors, pumps, and valves. Substitutes for asbestos include aramid fiber, aramid fiber in combination with graphite or polytetrafluoroethylene (PTFE), carbon fiber, glass fiber, and glass fiber impregnated with PTFE.

Composites. Asbestos and a range of other fibers are used in the production of thermosetting and thermoplastic composites that have important engineering applications. Although no single fiber type matches asbestos in all its properties, numerous substitutes are being introduced, including aramid fiber, glass fiber, carbon fiber, cotton, organic fibers, man-made mineral fibers, and particulate mineral fillers.

Heat-resistant textiles. Fibers woven into heat-resistant textiles must withstand temperatures of 200–1200°C, and in some cases molten metal splash, welding sparks, and naked flame. Asbestos has a service temperature of approximately 600°C. Refractory fibers are used at higher temperatures and synthetic organic fibers at lower temperatures. Various blends of organic, glass, metal, and synthetic fibers have also been developed for particular applications.

General Perspectives on Fiber Hazards

In this paper we are primarily concerned with fibrogenic and carcinogenic properties of fibers. It is generally accepted that to be pathogenic, fibers must be long, thin, and durable, although there is no consensus on the qualifying values of these parameters. Other factors such as fiber chemistry may also contribute to pathogenicity; this must be considered when deliberating the use of substitute fibers.

Fiber diameter is the major determinant of the falling speed in air, and hence of the fiber concentration where the aerosol is generated. The diameter also determines the probability that individual fibers will deposit in the alveolar region of the lung: mineral fibers larger than approximately 3 μm diameter are too large to reach the alveoli and deposition there is maximal at approximately 1 μm diameter, whereas in the ciliated airways these values are slightly larger. Because it critically effects both exposure concentrations and deposition, diameter should be considered a primary component of the intrinsic hazard of a fiber.

Once fibers have been inhaled, the integrated dose to the lung depends on their biopersistence, which is a function both of their durability and of lung clearance. Long fibers in the alveoli and nonciliated airways are removed only slowly by macrophages, so that durability is the principal determinant of their residence time. However, durability can be viewed in two ways. The first is resistance to dissolution in the lung, which is largely determined by the chemical composition of the fiber concerned. The second aspect of fiber durability is fragmentation (transverse breakage) in the lung. Mesothelioma induction requires a minimum fiber length. The minimum length is generally accepted as at least 8 μm although most workers would support a figure of 10 μm, and the true value may be closer to 20 μm (*5*). The induction of fibrosis is also more readily induced by long fibers. For pathogenicity in general, it is safe to assume that fibers < 4–5 μm in length are no more hazardous than the same material in nonfibrous form. Thus, if fibers fragment, they will be both less pathogenic and more readily removed.

The majority of fibrous products are produced in bulk form, often as wool or blanket, with staple lengths that may be measured in centimeters. Fiber dustiness, i.e., the ability of these products to fragment and release dust into the air, is an important determinant of hazard. It depends on the breakage rate of the filaments, which is a function of *a*) stiffness, *b*) resistance to shear, *c*) whether the fractures are propagated lengthways or transversely, and *d*) the respirability of the resulting fibrous dust. However, the effect of fiber geometry and composition on dustiness is not always easy to predict. Many composite materials bind fibers so that they cannot be released in normal use, although some may be released when the material is cut or abraded. Similarly, many fiber preparations contain binder or dust-suppression agents that inhibit fiber release.

In addition, in the case of fiber cement, such features as the weathering rate and its effect on the propensity to release fiber, the extent to which the composite may be used in a given situation or location, and whether the material is intended for indoor or outdoor use, will all have major consequences on both the exposure potential and the likely exposure rate.

Hazardous Properties of Chrysotile

The diseases associated with exposure to different forms of mineral fiber have been well characterized, although the mechanisms are still under investigation. The most important outcomes are diffuse interstitial fibrosis (asbestosis), lung cancer, and mesothelioma. Although it is not our intention to review all of the toxicologic properties and health effects of chrysotile, some observations are pertinent to the present comparison with substitute fibers.

Chrysotile is intrinsically hazardous because of its propensity to split longitudinally and produce thin respirable fibers (Figure 1). It is also moderately durable in the lung.

The carcinogenicity of chrysotile cannot be considered without taking into account the presence of varying concentrations of the fibrous amphibole tremolite, especially in the Canadian product. Few would dispute that tremolite is more carcinogenic than chrysotile on a weight-for-weight basis, partly or perhaps largely because of its greater durability. However, it has been claimed that mesotheliomas arising in asbestos workers are mainly attributable to tremolite (the amphibole hypothesis), and some would extend this to include lung tumors.

There is general agreement that chrysotile itself, whether contaminated with amphiboles or not, can cause lung cancer. Evidence for this comes from epidemiologic studies of workers who have been exposed to high levels of asbestos, and from studies on experimental animals that have used even higher dust concentrations.

One of the largest epidemiologic studies is that by McDonald and co-workers (*6*) on 11,000 Quebec miners and millers. This cohort study has recently been updated and is now essentially complete (*6*). It shows a clear excess of lung cancer in the three highest exposure groups. However, the 38 mesothelioma cases observed could not be related to exposure levels in the same way (*7*). From the latest analyses, the evidence relating to mesothelioma supports the amphibole hypothesis but cannot prove it. This is because we do not know the time-integrated lung doses of tremolite and chrysotile over the lifetimes of these workers and because epidemiologic evaluations provide proof of association but not cause.

A higher rate of lung cancer was found in the study of textile workers in Charleston, South Carolina (*8*). These workers were exposed to chrysotile from Quebec. The

excess lung cancer was best described by a dose–response relationship in which the relative risk increased linearly with exposure (*8*). The slope of the dose–response curve was over 10 times greater than the slope derived from the study of the Quebec workers, and this has been subject to various explanations. There were hardly any cases of mesothelioma, which, bearing in mind that most of the tremolite was removed during processing, supports the contention that this type of tumor is not caused by chrysotile.

Among asbestos-cement workers there have been some reported increases in the incidence of both lung cancer and mesothelioma, although at least some of these may be attributed to previous or concomitant crocidolite exposure. Most studies have not found increases in mesothelioma in either the asbestos-cement or the friction material industries [reviewed by Meldrum (*9*)].

The question of whether there is a threshold dose has not been settled by experimental work, any more than by epidemiologic studies. Results with intrapleural and intraperitoneal injection of mineral fibers are consistent with a threshold for the induction of mesothelioma. Much of the lung cancer data can be fitted by simple dose–response curves that do not include a threshold. However, there have been other experiments where chrysotile failed to produce any tumors even at high doses (*9*). The statistical requirements to demonstrate a threshold in a convincing manner are not easily met because very large numbers of animals are needed for the low-dose groups. This has been well illustrated in a study by Sanders et al. (*10*) for lung cancer induced by inhaled plutonium dioxide. In this study a threshold was clearly demonstrated, but only by using large numbers of animals (> 1,000) in both the zero-dose (control) group and the lowest dose group. No such study has been undertaken for asbestos or any other type of mineral fiber.

A number of epidemiologic studies of lung cancer have investigated whether there is interaction between asbestos exposure and cigarette smoking. Most of the studies involved populations exposed to amphiboles as well as chrysotile. The data are best described by a multiplicative (synergistic) interaction between asbestos and cigarette smoke (*11*). Rats that were injected with *N*-nitrosoheptamethyleneimine, a specific lung carcinogen, and inhaled chrysotile produced pulmonary tumors and hyperplastic responses in an apparently synergistic fashion (*12*). Unlike lung cancer, there appears to be no association between mesothelioma and cigarette smoking.

High doses of chrysotile are also both inflammatory and fibrogenic. It is generally agreed that asbestos will not cause cancer without prior chronic inflammation, but there is less consensus on whether this must progress to fibrosis. Although both asbestosis and lung cancer in humans can be induced by exposure to chrysotile, there is no agreement as to whether the two diseases run in parallel because of a common cause—inflammation (*13*)—or whether the development of frank fibrosis is a prerequisite for increased cancer incidence (*14*). In an extensive review, Henderson et al. (*15*) confirmed this lack of agreement, but noted a change in the balance of evidence in favor of the view that the fiber load itself is the main determinant for lung carcinogenesis. Asbestosis and lung cancer have broadly similar dose–response relationships, similar latent periods, and depend in the same way on fiber length [reviewed by Meldrum (*9*)].

Hazardous Properties of Substitute Fibers

It is important to realize that the volume of information available for the substitute fibers will always be less than that for chrysotile, especially regarding effects on humans. This is because of their relatively recent introduction and the fact that occupational exposures are not likely to match the high levels seen in the past with asbestos.

PVA fibers. The diameter of PVA fibers, as manufactured, is well above the respirable limit and most of them are not inhalable. They have a lower density (~ 1.3) as compared to mineral fibers, so that the respirable limit for PVA will be approximately 7 µm, versus 3 µm for mineral fibers. Nevertheless, the fibers are mostly in the range of 10–16 µm diameter. There is evidence that they do not fibrillate (split lengthwise). Many of the particles seen in the atmosphere are nonfibrous.

Although the published toxicologic information on PVA is relatively sparse, the parent material has been used extensively in surgery and has food contact clearance (*16*), presumably based on unpublished studies. Indications of an accumulation of oligomers in the kidney in some circumstances [e.g., Carver (*17*)] mean that the spectrum of molecular weight of material in the fibers as used should be considered, especially if a smaller diameter material were to be produced. The material will degrade only slowly, if at all, in the lungs.

Thus, substitution of PVA for asbestos fibers in products such as asbestos cement should result in reduced exposures. This prediction has been confirmed in industrial applications where very low fiber counts have been experienced. Misuse of installed material would not result in significant exposure.

Aramid fibers. Aramid fibers are also of predominantly coarse diameter (10–12 µm diameter as produced) and thus above the respirable limit, corrected for density, of 6–7 µm diameter. However, respirable fibrils of approximately 0.2 µm diameter are present on the surface of the fibers as produced, and can be liberated in operations with a high-energy input. The fibers do not fibrillate under pressure, although there is the potential to liberate fibrous wear fragments when shear forces are applied.

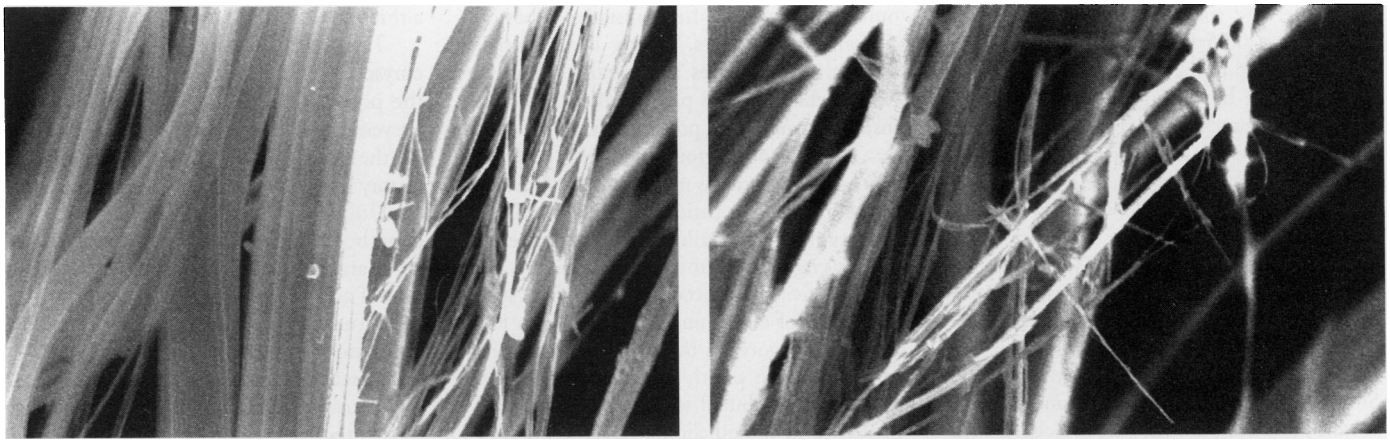

Figure 1. Chrysotile asbestos fibers seen by scanning electron microscopy. Note the fine, curly fibrils that are readily formed from chrysotile.

In recent studies *p*-aramid fibrils were less biopersistent overall than chrysotile in both rat and hamster lung (*18–21*). The longer aramid fibrils tended to fragment, unlike the longer chrysotile fibers that were therefore preferentially retained. High doses of *p*-aramid caused a small increase in lung cell proliferation, which disappeared by 5 days after the end of the exposure period in the rat, and by 1 month in the hamster. At the same level of exposure, chrysotile markedly increased cell proliferation in airway, alveolar, and subpleural tissue during 0–3 months postexposure (*20–22*).

Other animal experiments have shown fibrosis in response to high doses of aramid, but the associated proliferative keratinizing cysts are generally considered irrelevant for human risk assessment because there is no equivalent lesion in humans and because they occur in rats only at levels where lung clearance mechanisms are severely inhibited (*16*). Mesothelioma incidence in rats following intraperitoneal injection of fibrils is below the level normally considered positive, but some researchers consider there is a marginal effect. In this respect the fiber is no worse than chrysotile, where mesothelioma induction by pure fiber is at most a weak association.

On balance, the use of aramid fibers should result in reduced levels of fiber exposure as compared to chrysotile asbestos and the fibrils released will be no more toxic and will be less biopersistent. The predicted reduction in absolute exposure levels has been achieved in industrial practice. Misuse of installed material would not be expected to give significant exposures.

Cellulose fibers. Cellulose fibers are produced from a variety of natural sources and are reportedly predominantly nonrespirable, although experimental studies as well as industrial surveys have shown some potential to produce respirable fibers (*23*). The extent of fibrillation is not established but remains a possibility. In the UK fiber-cement industry the process is less dusty than processes using chrysotile, and the majority of fiber counts are less than 0.05 fibers/mL, although occasional peaks up to 0.2 fibers/mL may occur.

Cellulose has been used in the paper industry for hundreds of years with little evidence of disease, even at relatively high exposure levels. Although there is limited epidemiologic evidence of an increase in the lung cancer rate, smoking was not corrected for, so the etiology is uncertain (*24*). Wood dust has been associated with sinonasal cancer, but mainly for certain hardwoods; softwood was less potent or inactive, suggesting that the cellulose content was not the primary cause. Similarly, extensive reports of byssinosis in the cotton-processing industry are associated with contaminants rather than pure cellulose fiber.

Recent experimental evidence has shown that cellulose fiber is more biopersistent than chrysotile in the rat (*25*), but the lungs would have been overloaded by the high doses used, and clearance probably thereby impaired (*26*).

The toxicity of cellulose fibers has recently been reviewed (*24*). For a material with such wide application there are surprisingly few experimental data. The fibers were toxic to mouse macrophages *in vitro*, as shown by the release of lactic dehydrogenase. This was not confirmed subsequently with rat macrophages, although a high dose of cellulose did cause a transient inflammatory response *in vivo* (*27*). Cellulose fibers were as effective as chrysotile and crocidolite in stimulating macrophages to release inflammogenic substances such as interleukin-1, and were more effective than asbestos in stimulating the release of prostaglandins. In another recent study, cellulose instilled into rat lung produced a persistent granulomatous response (*28*), but again the high dose used would certainly have caused overload and thus inhibited normal clearance by macrophages. The inflammatory response to cellulose may also apply to nonfibrous material, although at low doses this will be removed more readily than long fibers by alveolar macrophages and mucociliary clearance.

On balance, the coarse fiber structure and the long experience in use indicate that substitution of cellulose fiber for chrysotile asbestos should result in reduced occupational exposures to fiber and lower levels of deposition in the lung. The apparent biopersistence of cellulose in the lung would be a possible cause for concern if the potential for limited lung damage is confirmed.

Exposure Levels

In UK industry, exposure to bulk chrysotile fiber is restricted to a small number of locations where the material is received and prepared for admixture with other components of the final product. The general public is exposed environmentally from geological outcrops, as well as from installed asbestos-containing products, mostly in buildings. The latter exposures are usually minimal, except where there is prolonged contact with installed materials in poor condition (*5*).

There are other groups for which potential exposure to asbestos is more difficult to define. These include occupationally exposed individuals, typically in building maintenance, or people engaged in home improvement as a leisure activity. Paraoccupational activity such as laundry of contaminated clothing also falls into this category. In the past, such exposures may have been poorly controlled, and have probably contributed to the current elevated incidence of mesothelioma in some building and maintenance trades (*29*).

For substitute fibers, the general considerations relating to exposures and potentially exposed groups are similar to those for chrysotile. The best practice in UK industry results in minimal fiber exposure levels in the workplace, especially for cellulose. Cellulose is available for supply to the cement industry as sheets or briquets, which are placed directly into water. PVA is imported and supplied in bales; fiber counts can be readily maintained below 0.05 fibers/mL. In friction product manufacture, substitute fibers are generally handled and monitored by the same practices developed for asbestos, and fiber counts kept below the same limits. If this is maintained, exposure to aramid fibers will be no greater than for asbestos, so that the resultant risk will be less.

Conclusions

There are now practicable substitutes for the major remaining uses of chrysotile. Although lack of a full health and toxicologic data set precludes a comprehensive assessment of the safety of substitute fibers, the application of basic principles of fiber toxicology enables a pragmatic decision to be made on the relative safety of potential substitutes. Our judgment is based on relative considerations of the intrinsic properties of fibers, on the pathogenicity of chrysotile in comparison with that of substitute fibers, and on the potential for uncontrollable exposures. The three parameters of dose, dimension (especially diameter), and durability are key to determining the differential hazards. Due consideration of these factors leads us to the following conclusions regarding chrysotile and its main substitutes.

Chrysotile per se can cause lung cancer and asbestosis; it is less clear that chrysotile alone can also cause mesothelioma in humans, and indeed it may not, whereas tremolite and other amphiboles certainly can do so. There is no definitive evidence for a threshold exposure level for lung cancer induction, although some studies suggest that a threshold does exist.

The intrinsic hazardous properties of chrysotile can never be "engineered out," and the potential for harm will always remain. Prevention of ill-health will thus always rely on the control of exposure, something that history has shown cannot be guaranteed.

Unlike chrysotile, substitute fibers can often be designed or selected to have particular characteristics. Criteria for the substitution of asbestos by other fibers include *a*) the substitute fibers are not in the respirable range, do not readily fibrillate, and/or are less durable than chrysotile; *b*) other materials that must be incorporated into the replacement product do not, in combination with the replacement fiber, produce more harm

overall than chrysotile alone; *c*) the replacement product has an equivalent or acceptable performance; and *d*) substitution would result in overall lower fiber exposures during manufacture and use and disposal, taking into account likely exposures. The same general principles can be applied to substitute fibers other than those considered here.

We judge that PVA fibers will pose less risk than chrysotile because they are generally too large to be respirable, do not fibrillate, and the parent material causes little or no tissue reaction. Aramid fibers have a reduced potential for exposure when compared to chrysotile because they are generally of high diameter and the production of respirable fibrils is energy intensive. The fibrils are less pathogenic than chrysotile, are less biopersistent, and are biodegradable. Cellulose has the benefit of long experience of use in a variety of industries without having raised significant concern. The potential for the generation of respirable fibers seems to be less than is the case for chrysotile, although fibrillation is possible. Cellulose is durable in the lung, and its biological properties should therefore be investigated further. However, exposure levels for current uses are low, and it is biodegradable in the environment.

We believe that the continued use of chrysotile in asbestos-cement products is not justifiable in the face of available and technically adequate substitutes. Likewise, there seems to be no justification for the continued residual use of chrysotile in friction materials.

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